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                  Substances (PICCS) has been added to CHEMLIST
 NEWS  3  Oct 27  New Extraction Code PAX now available in Derwent
                  Files
 NEWS  4  Oct 27  SET ABBREVIATIONS and SET PLURALS extended in
                  Derwent World Patents Index files
 NEWS  5  Oct 27  Patent Assignee Code Dictionary now available
                  in Derwent Patent Files
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FILE 'HOME' ENTERED AT 13:06:46 ON 01 DEC 2000

=> file biosis, medline, uspat, wpids

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FILE 'BIOSIS' ENTERED AT 13:09:16 ON 01 DEC 2000
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FILE 'USPATFULL' ENTERED AT 13:09:16 ON 01 DEC 2000
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=> s FGF-2

L1 2101 FGF-2

=> s bFGF or basic fibroblast growth factor

L2 16989 BFGF OR BASIC FIBROBLAST GROWTH FACTOR

=> s acidic fibroblast growth factor

L3 2339 ACIDIC FIBROBLAST GROWTH FACTOR

=> s l1 and l2

L4 648 L1 AND L2

=> s l3 and l4

L5 59 L3 AND L4

=> s l5 and human

L6 45 L5 AND HUMAN

=> s l6 and angiogenesis

L7 15 L6 AND ANGIOGENESIS

=> d l7 ti abs ibib tot

L7 ANSWER 1 OF 15 BIOSIS COPYRIGHT 2000 BIOSIS
TI INACTIVATION OF **HUMAN** FIBROBLAST GROWTH FACTOR-1 FGF-1 ACTIVITY BY INTERACTION WITH COPPER IONS INVOLVES FGF-1 DIMER FORMATION INDUCED BY COPPER-CATALYZED OXIDATION.
AB Although the angiogenic proteins **acidic fibroblast growth factor** (FGF-1) and **basic fibroblast growth factor** (FGF-I) and **basic fibroblast growth factor** (**FGF-2**) both interact with the transition metal copper, itself a putative modulator of **angiogenesis**, a role for copper in FGF function has not been established. Using nonreducing sodium dodecyl sulfate polyacrylamide gel electrophoresis, we detect the complete conversion of recombinant forms of **human** FGF-1 monomer protein to FGF-1 homodimers after exposure to copper ions. In contrast, not all forms of bovine FGF-1 isolated from bovine brain or a recombinant preparation of **human FGF-2** completely formed homodimers after exposure to copper ions under similar conditons. Since the copper-induced FGF-1 homodimers reverted to the monomer form in the presence of dithiothreitol, specific alkylation of cysteine residues by pyridylethylation prevented FGF-1 homodimer formation, and preformed FGF-1 homodimers could not be dissociated by the metal chelator EDTA, FGF-1 dimer formation appeared to result from the formation of intermolecular disulfide bonds by copper-induced oxidation of sulfhydryl residues. FGF-1 homodimers bound with similar apparent affinity as FGF-1 monomers to

immobilized copper ions, both eluting at 60 mM imidazole. Both **human** FGF-1 monomer and dimer forms had a 6-fold higher apparent affinity for immobilized copper ions, as compared with **human FGF-2**, which eluted in the monomer form at 10 mM imidazole. Further, in contrast to FGF-1 monomers, which dissociate from immobilized heparin in 1.0 M NaCl, preformed FGF-1 homodimers had reduced apparent affinity for immobilized heparin and eluted at 0.4 M NaCl. In contrast, the apparent affinity of **human FGF-2** for immobilized heparin was unaffected after exposure to copper ions. Heparin appeared to modulate the formatin of copper-induced intermolecular disulfide bonds for FGF-1 but not **FGF-2**, since co-incubation of heparin and copper with FGF-1 monomers resulted in dimers and other oligomeric complexes. FGF-1 copper-induced homodimers failed to induce mitogenesis in [3H]thymidine incorporation assays, an effect which could be reversed by treatment with dithiothreitol, whereas **FGF-2**-induced mitogenic activity was relatively unaffected by pretreatment with copper. The differences between **human** FGF-1 and **FGF-2** in protein-copper interactions may be due to differing free thiol content and arrangement between the two proteins. A recombinant **human** FGF-1 mutant containing the two cysteines conserved throughout the FGF family of proteins but lacking a cysteine residue (Cys 131) present in wild-type **human** FGF-1 but not **human FGF-2** readily formed copper-induced dimers. This suggests FGF-1-copper interactions may involve the cysteine residues converved among all the members of the GFG family. Despite the differences in copper interactions between FGF-1 and **FGF-2**, copper-induced heterodimers between these two proteins were demonstrated, indicating FGF-1 and **FGF-2** can form mixed thiols with each other. The difference in copper interactions between different forms of FGF-1 and **FGF-2** are important in further attempts to determine any putative role for copper in FGF action.

ACCESSION NUMBER: 1992:344929 BIOSIS
DOCUMENT NUMBER: BA94:37154
TITLE: INACTIVATION OF **HUMAN** FIBROBLAST GROWTH FACTOR-1 FGF-1 ACTIVITY BY INTERACTION WITH COPPER IONS INVOLVES FGF-1 DIMER FORMATION INDUCED BY COPPER-CATALYZED OXIDATION.
AUTHOR(S): ENGLEKA K A; MACIAG T
CORPORATE SOURCE: DEP. MOLECULAR BIOLOGY, HOLLAND LABORATORY, AMERICAN RED CROSS, 15601 CRABBS BRANCH WAY, ROCKVILLE, MD. 20855.
SOURCE: J BIOL CHEM, (1992) 267 (16), 11307-11315.
CODEN: JBCHA3. ISSN: 0021-9258.
FILE SEGMENT: BA; OLD
LANGUAGE: English

L7 ANSWER 2 OF 15 MEDLINE
TI Protection of rat myocardium by mitogenic and non-mitogenic fibroblast growth factor during post-ischemic reperfusion.
AB The effects of **acidic fibroblast growth factor** (FGF-1) and **basic fibroblast growth factor** (**FGF-2**) and a non mitogenic form of FGF1 on myocardial ischemia and reperfusion were assessed. Rats underwent 10 minutes of coronary artery occlusion followed by 24 hours of reperfusion. Creatinine kinase content of the affected myocardium showed that both fibroblast growth factors 1 and 2 effectively protected against ischemia reperfusion injury ($p < 0.01$), and that the vasoactive but nonmitogenic form of the FGF1 was equally protective ($p < 0.01$ versus control + vehicle). The results were confirmed by light and electron-microscopy histological studies. Histological evaluations after treatment with the non-mitogenic fibroblast growth factor 1 showed that it

did not generate the severe hyperplasia and connective tissue disorganization observed with the native mitogenic proteins. The possibility of using a non-mitogenic form of fibroblast growth factor for cardio-protection circumvents many of the potentially undesirable effects that may derive from systemically introducing broad spectrum acting fibroblast growth factors in vivo. This myocardial protection observed 24 hours after the treatment with fibroblast growth factors, and the efficacy of the non-mitogenic form of the protein, also suggest that the protective effect of fibroblast growth factors may be due to the increased blood flow rather than to **angiogenesis**.

ACCESSION NUMBER: 1998065378 MEDLINE
DOCUMENT NUMBER: 98065378
TITLE: Protection of rat myocardium by mitogenic and non-mitogenic fibroblast growth factor during post-ischemic reperfusion.
AUTHOR: Cuevas P; Carceller F; Lozano R M; Crespo A; Zazo M; Gimenez-Gallego G
CORPORATE SOURCE: Hospital Universitario Ramon y Cajal, Madrid, Spain.
SOURCE: GROWTH FACTORS, (1997) 15 (1) 29-40.
Journal code: AOI. ISSN: 0897-7194.
PUB. COUNTRY: Switzerland
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199803
ENTRY WEEK: 19980303

L7 ANSWER 3 OF 15 MEDLINE
TI Fibroblast growth factors: at the heart of **angiogenesis**.
AB **Acidic fibroblast growth factor** (FGF-1) and **basic fibroblast growth factor** (**FGF-2**) are ubiquitous cytokines found in many tissues. They have effects on multiple cell types derived from mesoderm and neuroectoderm, including endothelial cells. In this review the structure and function of the fibroblast growth factor family and its receptors are described. The evidence implicating both FGF-1 and **FGF-2** in the control of blood vessel formation is presented and their involvement in normal and pathological **angiogenesis** during adult life is then described in more detail.

ACCESSION NUMBER: 95368012 MEDLINE
DOCUMENT NUMBER: 95368012
TITLE: Fibroblast growth factors: at the heart of **angiogenesis**.
AUTHOR: Slavin J
CORPORATE SOURCE: Department of Surgery, Aintree Hospitals, Liverpool..
SOURCE: CELL BIOLOGY INTERNATIONAL, (1995 May) 19 (5) 431-44.
Ref: 101
Journal code: BPN. ISSN: 1065-6995.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199511

L7 ANSWER 4 OF 15 MEDLINE
TI Inactivation of **human** fibroblast growth factor-1 (FGF-1)

activity by interaction with copper ions involves FGF-1 dimer formation induced by copper-catalyzed oxidation.

AB Although the angiogenic proteins **acidic fibroblast growth factor** (FGF-1) and **basic fibroblast growth factor** (**FGF-2**) both interact with the transition metal copper, itself a putative modulator of **angiogenesis**, a role for copper in FGF function has not been established. Using nonreducing sodium dodecyl sulfate-polyacrylamide gel electrophoresis, we detect the complete conversion of recombinant forms of **human** FGF-1 monomer protein to FGF-1 homodimers after exposure to copper ions. In contrast, not all forms of bovine FGF-1 isolated from bovine brain or a recombinant preparation of **human FGF-2** completely formed homodimers after exposure to copper ions under similar conditions. Since the copper-induced FGF-1 homodimers reverted to the monomer form in the presence of dithiothreitol, specific alkylation of cysteine residues by pyridylethylation prevented FGF-1 homodimer formation, and preformed FGF-1 homodimers could not be dissociated by the metal chelator EDTA, FGF-1 dimer formation appeared to result from the formation of intermolecular disulfide bonds by copper-induced oxidation of sulfhydryl residues. FGF-1 homodimers bound with similar apparent affinity as FGF-1 monomers to immobilized copper ions, both eluting at 60 mM imidazole. Both **human** FGF-1 monomer and dimer forms had a 6-fold higher apparent affinity for immobilized copper ions, as compared with **human FGF-2**, which eluted in the monomer form at 10 mM imidazole. Further, in contrast to FGF-1 monomers, which dissociate from immobilized heparin in 1.0 M NaCl, preformed FGF-1 homodimers had reduced apparent affinity for immobilized heparin and eluted at 0.4 M NaCl. In contrast, the apparent affinity of **human FGF-2** for immobilized heparin was unaffected after exposure to copper ions. Heparin appeared to modulate the formation of copper-induced intermolecular disulfide bonds for FGF-1 but not **FGF-2**, since co-incubation of heparin and copper with FGF-1 monomers resulted in dimers and other oligomeric complexes. FGF-1 copper-induced homodimers failed to induce mitogenesis in [3H]thymidine incorporation assays, an effect which could be reversed by treatment with dithiothreitol, whereas **FGF-2**-induced mitogenic activity was relatively unaffected by pretreatment with copper. The differences between **human** FGF-1 and **FGF-2** in protein-copper interactions may be due to differing free thiol content and arrangement between the two proteins. A recombinant **human** FGF-1 mutant containing the two cysteines conserved throughout the FGF family of proteins but lacking a cysteine residue (Cys 131) present in wild-type **human** FGF-1 but not **human FGF-2** readily formed copper-induced dimers.(ABSTRACT TRUNCATED AT 400 WORDS)

ACCESSION NUMBER: 92283839 MEDLINE
DOCUMENT NUMBER: 92283839
TITLE: Inactivation of **human** fibroblast growth factor-1 (FGF-1) activity by interaction with copper ions involves FGF-1 dimer formation induced by copper-catalyzed oxidation.
AUTHOR: Engleka K A; Maciag T
CORPORATE SOURCE: Department of Molecular Biology, Holland Laboratory, American Red Cross, Rockville, Maryland 20855.
CONTRACT NUMBER: HL32348 (NHLBI)
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1992 Jun 5) 267 (16) 11307-15.
Journal code: HIV. ISSN: 0021-9258.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; Cancer Journals

ENTRY MONTH: 199209

L7 ANSWER 5 OF 15 USPATFULL
TI Chitin hydrogels, methods of their production and use
AB This invention is directed to the preparation and utilization of supplemented chitin hydrogels, such as chitosan hydrogels. Further provided are biomaterials comprising same. The particular supplement delivered by the chitin hydrogel is selected as a function of its intended use. In one embodiment, this invention provides a composition of matter, comprising a chitin hydrogel or chitin-derived hydrogel, wherein the hydrogel does not inhibit full-thickness skin wound healing.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2000:128306 USPATFULL
TITLE:	Chitin hydrogels, methods of their production and use
INVENTOR(S):	Drohan, William N., Springfield, VA, United States MacPhee, Martin J., Gaithersburg, MD, United States Miekka, Shirley I., Gaithersburg, MD, United States Singh, Manish S., Columbia, MD, United States Elson, Clive, Halifax, Canada Taylor, Jr., John R., New York, NY, United States
PATENT ASSIGNEE(S):	Chitogenics, Inc., Morristown, NJ, United States (U.S. corporation) The American National Red Cross, Washington, DC, United States (U.S. corporation) Coalition for Hemophilia B, New York, NY, United States (U.S. corporation)

	NUMBER	DATE
PATENT INFORMATION:	US 6124273	20000926
APPLICATION INFO.:	US 1997-960555	19971013 (8)
RELATED APPLN. INFO.:	Continuation of Ser. No. US 1996-659999, filed on 7 Jun 1996, now abandoned	
PRIORITY INFORMATION:	US 1995-109	19950609 (60)

DOCUMENT TYPE:	Utility
PRIMARY EXAMINER:	Fonda, Kathleen K.
LEGAL REPRESENTATIVE:	Lahive & Cockfield, LLP
NUMBER OF CLAIMS:	32
EXEMPLARY CLAIM:	1
NUMBER OF DRAWINGS:	6 Drawing Figure(s); 3 Drawing Page(s)
LINE COUNT:	2441

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 6 OF 15 USPATFULL
TI Supplemented and unsupplemented tissue sealants, method of their production and use
AB This invention provides supplemented tissue sealants, methods for their production and use thereof. Disclosed are tissue sealants supplemented with at least one cytotoxin or cell proliferation inhibiting composition. The composition may be further supplemented with, for example, one or more antibodies, analgesics, anticoagulants, anti-inflammatory compounds, antimicrobial compositions, cytokines, drugs, growth factors, interferons, hormones, lipids, demineralized bone or bone morphogenetic proteins, cartilage inducing factors,

oligonucleotides polymers, polysaccharides, polypeptides, protease inhibitors, vasoconstrictors or vasodilators, vitamins, minerals, stabilizers and the like.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2000:121069 USPATFULL
TITLE:	Supplemented and unsupplemented tissue sealants, method of their production and use
INVENTOR(S):	MacPhee, Martin James, Gaithersburg, MD, United States Drohan, William Nash, Springfield, VA, United States Liau, Gene, Darnestown, MD, United States Haudenschild, Christian, Rockville, MD, United States
PATENT ASSIGNEE(S):	The American National Red Cross, Falls Church, VA, United States (U.S. corporation)

	NUMBER	DATE
PATENT INFORMATION:	US 6117425	20000912
APPLICATION INFO.:	US 1995-474086	19950607 (8)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1994-351006, filed on 7 Dec 1994, now abandoned which is a continuation-in-part of Ser. No. US 1994-328552, filed on 25 Oct 1994, now abandoned which is a continuation of Ser. No. US 1993-31164, filed on 12 Mar 1993, now abandoned which is a continuation-in-part of Ser. No. US 1990-618419, filed on 27 Nov 1990, now abandoned which is a continuation-in-part of Ser. No. US 1991-798919, filed on 27 Nov 1991, now abandoned

DOCUMENT TYPE:	Utility
PRIMARY EXAMINER:	Woodward, M Patrick
ASSISTANT EXAMINER:	Zeman, Mary K
LEGAL REPRESENTATIVE:	Sterne, Kessler Goldstein & Fox P.L.L.C.
NUMBER OF CLAIMS:	57
EXEMPLARY CLAIM:	1,2,3
NUMBER OF DRAWINGS:	53 Drawing Figure(s); 36 Drawing Page(s)
LINE COUNT:	4910

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 7 OF 15 USPATFULL

TI Keratinocyte growth factor-2

AB This invention relates to newly identified polynucleotides, polypeptides encoded by such polynucleotides, the use of such polynucleotides and polypeptides, as well as the production of such polynucleotides and polypeptides. More particularly, the polypeptide of the present invention is a Keratinocyte Growth Factor, sometimes hereinafter referred to as "KGF-2" also formerly known as Fibroblast Growth Factor 12 (FGF-12). This invention further relates to the therapeutic use of KGF-2 to promote or accelerate wound healing. This invention also relates to novel mutant forms of KGF-2 that show enhanced activity, increased stability, higher yield or better solubility.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2000:77208 USPATFULL
TITLE:	Keratinocyte growth factor-2
INVENTOR(S):	Ruben, Steven M., Olney, MD, United States Jimenez, Pablo, Ellicott City, MD, United States Duan, D. Roxanne, Bethesda, MD, United States Rampy, Mark A., Gaithersburg, MD, United States Mendrick, Donna, Mt. Airy, MD, United States Zhang, Jun, Bethesda, MD, United States Ni, Jian, Rockville, MD, United States

	Moore, Paul A., Germantown, MD, United States Coleman, Timothy A., Gaithersburg, MD, United States Gruber, Joachim R., Chestnut Hill, MA, United States Dillon, Patrick J., Carlsbad, CA, United States Gentz, Reiner L., Rockville, MD, United States
PATENT ASSIGNEE(S):	Human Genome Sciences, Inc., Rockville, MD, United States (U.S. corporation)

	NUMBER	DATE
PATENT INFORMATION:	US 6077692	20000620
APPLICATION INFO.:	US 1998-23082	19980213 (9)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1997-910875, filed on 13 Aug 1997, now abandoned And a continuation-in-part of Ser. No. US 1997-862432, filed on 23 May 1997, now abandoned which is a division of Ser. No. US 1995-461195, filed on 5 Jun 1995, now abandoned which is a continuation-in-part of Ser. No. WO 1995-US1790, filed on 14 Feb 1995	

	NUMBER	DATE
PRIORITY INFORMATION:	US 1997-55561	19970813 (60)
	US 1997-39045	19970228 (60)
	US 1996-23852	19960813 (60)
	US 1997-68493	19971222 (60)

DOCUMENT TYPE:	Utility
PRIMARY EXAMINER:	Ulm, John
ASSISTANT EXAMINER:	Saoud, Christine
LEGAL REPRESENTATIVE:	Sterne, Kessler, Goldstein & Fox, P.L.L.C.
NUMBER OF CLAIMS:	683
EXEMPLARY CLAIM:	1
NUMBER OF DRAWINGS:	80 Drawing Figure(s); 64 Drawing Page(s)
LINE COUNT:	9626

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 8 OF 15 USPATFULL

TI Supplemented and unsupplemented tissue sealants, methods of their production and use

AB This invention provides a fibrin sealant dressing, wherein said fibrin sealant may be supplemented with at least one composition selected from,

for example, one or more regulatory compounds, antibody, antimicrobial compositions, analgesics, anticoagulants, antiproliferatives, anti-inflammatory compounds, cytokines, cytotoxins, drugs, growth factors, interferons, hormones, lipids, demineralized bone or bone morphogenetic proteins, cartilage inducing factors, oligonucleotides polymers, polysaccharides, polypeptides, protease inhibitors, vasoconstrictors or vasodilators, vitamins, minerals, stabilizers and the like. Also disclosed are methods of preparing and/or using the unsupplemented or supplemented fibrin sealant dressing.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2000:50372 USPATFULL
TITLE:	Supplemented and unsupplemented tissue sealants, methods of their production and use
INVENTOR(S):	MacPhee, Martin James, Gaithersburg, MD, United States Drohan, William Nash, Springfield, VA, United States Woolverton, Christoper J., Kent, OH, United States
PATENT ASSIGNEE(S):	The American National Red Cross, Washington, DC, United States (U.S. government)

	NUMBER	DATE
PATENT INFORMATION:	US 6054122	20000425
APPLICATION INFO.:	US 1995-479034	19950607 (8)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1994-351006, filed on 7 Dec 1994, now abandoned which is a continuation-in-part of Ser. No. US 1994-328552, filed on 25 Oct 1994, now abandoned which is a continuation of Ser. No. US 1993-31164, filed on 12 Mar 1993, now abandoned which is a continuation-in-part of Ser. No. US 1990-618419, filed on 27 Nov 1990, now abandoned

And

a continuation-in-part of Ser. No. US 1991-798919, filed on 27 Nov 1991, now abandoned

DOCUMENT TYPE: Utility
PRIMARY EXAMINER: Smith, Lynette F.
ASSISTANT EXAMINER: Zeman, Mary K
LEGAL REPRESENTATIVE: Sterne, Kessler, Goldstein & Fox P.L.L.C.
NUMBER OF CLAIMS: 43
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 50 Drawing Figure(s); 36 Drawing Page(s)
LINE COUNT: 4855
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 9 OF 15 USPATFULL

TI Method of cloning bovines using reprogrammed non-embryonic bovine cells

AB The present invention relates to cloning technologies. The invention relates in part to immortalized and totipotent cells useful for cloning animals, the embryos produced from these cells using nuclear transfer techniques, animals that arise from these cells and embryos, and materials, methods, and processes for establishing such cells, embryos, and animals.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2000:2039 USPATFULL
TITLE: Method of cloning bovines using reprogrammed non-embryonic bovine cells
INVENTOR(S): Strelchenko, Nikolai S., DeForest, WI, United States
Betthauser, Jeffrey M., Windsor, WI, United States
Jurgella, Gail L., Madison, WI, United States
Pace, Marvin M., DeForest, WI, United States
Bishop, Michael D., Rio, WI, United States
PATENT ASSIGNEE(S): Infigen, Inc., Deforest, WI, United States (U.S. corporation)

	NUMBER	DATE
PATENT INFORMATION:	US 6011197	20000104
APPLICATION INFO.:	US 1999-239922	19990128 (9)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1997-812851, filed on 6 Mar 1997 which is a continuation-in-part of Ser. No. US 812031

	NUMBER	DATE
PRIORITY INFORMATION:	US 1998-73019	19980129 (60)

DOCUMENT TYPE: Utility
PRIMARY EXAMINER: Crouch, Deborah
LEGAL REPRESENTATIVE: Lyon & Lyon LLP
NUMBER OF CLAIMS: 29
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 4 Drawing Figure(s); 4 Drawing Page(s)
LINE COUNT: 3520

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 10 OF 15 USPATFULL
TI Attenuation of wound healing processes
AB Glycosaminoglycans, including heparinases 1, 2 and 3 as well as chondroitinases AC and B from the Gram negative bacteria Flavobacterium heparinum, can be used either separately or in combination to manipulate cell proliferation. In one embodiment, heparinases are administered to degrade heparan sulfate components of the extracellular matrix, thereby allowing the heparin binding growth factors which are stored in the extracellular matrix to migrate to adjacent cells. The mobility of chemoattractant agents, growth factors and cells also can be increased by treating tissues with glycosaminoglycan degrading enzymes, both chondroitinases and heparinases. The enzymatic removal of chondroitin sulfates from cell surfaces effectively increases the availability of growth factor receptors on the cell's surface. Selectively removing heparan sulfate from cell surfaces while leaving the extracellular matrix intact, conversely, inhibits cell proliferation by down regulating the cell's response to growth factors. This is achieved by targeting heparin or heparan sulfate degrading activities to the cell surface. Targeting the heparin degrading activity can be achieved by genetically engineering a ligand binding functionality into the heparinase proteins, or by physically controlling the localized enzyme concentration through the method of administration.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	1999:159480 USPATFULL	
TITLE:	Attenuation of wound healing processes	
INVENTOR(S):	Zimmermann, Joseph, Elm Grove, WI, United States Vlodavsky, Israel, Jerusalem, Israel Bennett, Clark, Pierrefonds, Canada Danagher, Pamela, Montreal, Canada Broughton, Richard, Montreal, Canada	
PATENT ASSIGNEE(S):	Ibex Technologies R and D, Inc., Montreal, Canada (non-U.S. corporation)	
	NUMBER	DATE
PATENT INFORMATION:	US 5997863	19991207
APPLICATION INFO.:	US 1994-273109	19940708 (8)
DOCUMENT TYPE:	Utility	
PRIMARY EXAMINER:	Hobbs, Lisa J.	
LEGAL REPRESENTATIVE:	Arnall Golden & Gregory, LLP	
NUMBER OF CLAIMS:	17	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	9 Drawing Figure(s); 4 Drawing Page(s)	
LINE COUNT:	1299	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 11 OF 15 USPATFULL
TI Analogs for specific oligosaccharide-protein interactions and uses therefor
AB Disclosed are (1) methods for identifying natural and synthetic sequences having binding specificity for glycan-binding proteins, including proteins that act as effectors of biological activity, (2) compositions and methods of producing protein-specific glycosaminoglycan sequence and ligand antagonists capable of modulating the effector function of these ligands, and therapeutic compositions comprising these antagonists; and 3) compositions and methods for producing protein-specific glycosaminoglycan sequence analogs useful as agonists,

and therapeutic compositions comprising these agonists.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	1998:98884 USPATFULL
TITLE:	Analogs for specific oligosaccharide-protein interactions and uses therefor
INVENTOR(S):	Witt, Daniel P., Hamilton, MA, United States Herlihy, Jr., Walter C., Beverly, MA, United States
PATENT ASSIGNEE(S):	Repligen Corporation, Needham, MA, United States (U.S. corporation)

	NUMBER	DATE
PATENT INFORMATION:	US 5795860	19980818
APPLICATION INFO.:	US 1994-202989	19940228 (8)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1993-24558, filed on 1 Mar 1993, now abandoned	
DOCUMENT TYPE:	Utility	
PRIMARY EXAMINER:	Peselev, Elli	
LEGAL REPRESENTATIVE:	Testa, Hurwitz & Thibeault, LLP	
NUMBER OF CLAIMS:	67	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	23 Drawing Figure(s); 16 Drawing Page(s)	
LINE COUNT:	2449	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 12 OF 15 USPATFULL

TI Method and compositions of a bioartificial kidney suitable for use in vivo or ex vivo

AB A novel cell seeded hollow fiber bioreactor is described as a potential bioartificial kidney. Renal cells are seeded along a hollow fiber in a perfused bioreactor to reproduce the ultrafiltration function and transport function of the kidney. Maintenance of tissue specific function and ultrastructure suggest that this bioreactor provides an economical device for treating renal failure as well as studying renal tubululogenesis in vitro.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	97:104321 USPATFULL
TITLE:	Method and compositions of a bioartificial kidney suitable for use in vivo or ex vivo
INVENTOR(S):	Humes, H. David, Ann Arbor, MI, United States Cieslinski, Deborah A., Ann Arbor, MI, United States
PATENT ASSIGNEE(S):	The University of Michigan, Ann Arbor, MI, United States (U.S. corporation)

	NUMBER	DATE
PATENT INFORMATION:	US 5686289	19971111
APPLICATION INFO.:	US 1995-487327	19950607 (8)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1993-133436, filed on 8 Oct 1993	
DOCUMENT TYPE:	Utility	
PRIMARY EXAMINER:	Dodson, Shelley A.	
LEGAL REPRESENTATIVE:	Oblon, Spivak, McClelland, Maier & Neustadt, P.C.	
NUMBER OF CLAIMS:	8	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	25 Drawing Figure(s); 15 Drawing Page(s)	
LINE COUNT:	1372	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 13 OF 15 USPATFULL

TI Fibroblast growth factor conjugates

AB The invention provides a conjugate comprising FGF or other polypeptide reactive with an FGF receptor, and a cytotoxic agent. The cytotoxic agent can be a ribosome-inactivating protein (RIP), such as saporin, although other cytotoxic agents can also be advantageously used. The cytotoxic agent can be attached to FGF through a chemical bond, or the composition can be prepared as a chimera using techniques of recombinant DNA. The conjugate can be used to treat FGF-mediated pathophysiological conditions by specifically targeting cells having FGF receptors and inhibiting proliferation of or causing death of such cells. Additionally, the conjugate can be used to target cytotoxic agents into cells having FGF receptors to inhibit the proliferation of such cells. The conjugate can be purified on an immobilized-heparin column.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	97:96833 USPATFULL	
TITLE:	Fibroblast growth factor conjugates	
INVENTOR(S):	Lappi, Douglas A., Del Mar, CA, United States Baird, J. Andrew, San Diego, CA, United States	
PATENT ASSIGNEE(S):	The Salk Institute For Biological Studies, San Diego, CA, United States (U.S. corporation)	
	NUMBER	DATE
PATENT INFORMATION:	US 5679637	19971021
APPLICATION INFO.:	US 1995-463996	19950605 (8)
RELATED APPLN. INFO.:	Division of Ser. No. US 1994-257958, filed on 10 Jun 1994, now patented, Pat. No. US 5576288 which is a continuation of Ser. No. US 1993-24682, filed on 1 Mar 1993, now abandoned which is a continuation-in-part of Ser. No. US 1989-344109, filed on 27 Apr 1989, now patented, Pat. No. US 5191067	
DOCUMENT TYPE:	Utility	
PRIMARY EXAMINER:	Sayala, Chhaya D.	
LEGAL REPRESENTATIVE:	Fitch, Even, Tabin & Flannery	
NUMBER OF CLAIMS:	6	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	7 Drawing Figure(s); 6 Drawing Page(s)	
LINE COUNT:	576	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 14 OF 15 USPATFULL

TI Fibroblast growth factor conjugates

AB The invention provides a conjugate comprising FGF or other polypeptide reactive with an FGF receptor, and a cytotoxic agent. The cytotoxic agent can be a ribosome-inactivating protein (RIP), such as saporin, although other cytotoxic agents can also be advantageously used. The cytotoxic agent can be attached to FGF through a chemical bond, or the composition can be prepared as a chimera using techniques of recombinant DNA. The conjugate can be used to treat FGF-mediated pathophysiological conditions by specifically targeting cells having FGF receptors and inhibiting proliferation of or causing death of such cells. Additionally, the conjugate can be used to target cytotoxic agents into cells having FGF receptors to inhibit the proliferation of such cells. The conjugate can be purified on an immobilized-heparin column.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	96:106461 USPATFULL
TITLE:	Fibroblast growth factor conjugates
INVENTOR(S):	Lappi, Douglas A., Del Mar, CA, United States Baird, J. Andrew, San Diego, CA, United States
PATENT ASSIGNEE(S):	The Salk Institute For Biological Studies, La Jolla,

CA, United States (U.S. corporation)

	NUMBER	DATE
PATENT INFORMATION:	US 5576288	19961119
APPLICATION INFO.:	US 1994-257958	19940610 (8)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1993-24682, filed on 1 Mar 1993, now abandoned which is a continuation-in-part of Ser. No. US 1989-344109, filed on 27 Apr 1989, now patented, Pat. No. US 5191067
DOCUMENT TYPE: Utility
PRIMARY EXAMINER: Walsh, Stephen G.
LEGAL REPRESENTATIVE: Fitch, Even, Tabin & Flannery
NUMBER OF CLAIMS: 9
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 7 Drawing Figure(s); 6 Drawing Page(s)
LINE COUNT: 684
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 15 OF 15 USPATFULL
TI Methods and compositions of a bioartificial kidney suitable for use in vivo or ex vivo
AB A novel cell seeded hollow fiber bioreactor is described as a potential bioartificial kidney. Renal cells are seeded along a hollow fiber in a perfused bioreactor to reproduce the ultrafiltration function and transport function of the kidney. Maintenance of tissue specific function and ultrastructure suggest that this bioreactor provides an economical device for treating renal failure as well as studying renal tubululogenesis in vitro.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 96:77334 USPATFULL
TITLE: Methods and compositions of a bioartificial kidney suitable for use in vivo or ex vivo
INVENTOR(S): Humes, H. David, Ann Arbor, MI, United States
Cieslinski, Deborah A., Ann Arbor, MI, United States
PATENT ASSIGNEE(S): The Regents of The University of Michigan, Ann Arbor, MI, United States (U.S. corporation)

	NUMBER	DATE
PATENT INFORMATION:	US 5549674	19960827
APPLICATION INFO.:	US 1993-133436	19931008 (8)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1992-844758, filed on 2 Mar 1992, now patented, Pat. No. US 5429938
DOCUMENT TYPE: Utility
PRIMARY EXAMINER: Dodson, Shelley A.
LEGAL REPRESENTATIVE: Oblon, Spivak, McClelland, Maier, & Neustadt, P.C.
NUMBER OF CLAIMS: 59
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 25 Drawing Figure(s); 15 Drawing Page(s)
LINE COUNT: 1648
CAS INDEXING IS AVAILABLE FOR THIS PATENT.